Allergy Abstracts

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Asthma and Hay Fever

Intermittent Positive-Pressure Breathing. Appraisal of Use in Bronchodilator Therapy of Pulmonary Emphysema. Leslie, A., Dantes, D. A., and Rosove, L.: J. A. M. A. **160**: 125, 1956.

The effectiveness of intermittent positive-pressure breathing as an adjunct to bronchodilator therapy was studied in 33 patients with moderate to advanced pulmonary emphysema associated with various degrees of bronchospasm, fibrosis, bronchiectasis and chronic bronchopulmonary infection. In the first phase of the study, 3 bronchodilator drugs were power-nebulized with oxygen for inhalation and were given in random sequence so as to determine the most effective drug for each patient. The appropriate drug was then used four times for each patient, twice with and twice without added intermittent positive pressure of 15 to 20 cm. H_2O. The following pulmonary functions were measured before and again 10 minutes after each treatment: volume of deep respiration, inspiratory and expiratory vital capacities, and maximum exhalation differences. In later phases of the study, treatments were given

twice daily for 20 minutes for 4 or 8 weeks, again with and without pressure breathing. Although 6 of the 33 cases showed better therapeutic responses with intermittent positive-pressure breathing than with power nebulization alone, it was concluded that the effect of the power nebulization of bronchodilator agents is not, as a rule, enhanced by this procedure. M. C.

Severe Toxic Effects From Aminophylline and Theophylline Suppositories in Children. Nolke, A. C.: J. A. M. A. **161**: 693, 1956.

Severe toxic reactions following the use of aminophylline or theophylline suppositories occurred in 13 children, 11 of whom were asthmatics. There were 4 deaths. Early symptoms of toxicity were anorexia, nausea, and irritability which were followed by vomiting and agitated maniacal behavior. In the end stages, there ensued a state of delirium and coma with convulsions, hyperthermia, profuse diaphoresis, and finally vasomotor collapse. The eldest patient in this series was 6 years of age. Ten patients were less than 3 years of age. Toxic symptoms occurred after the first suppository in 4 patients and after the second in 10 cases. The majority of the patients received doses of the suppositories which were regarded to be within the therapeutic range and not excessive. Treatment in all instances was symptomatic and the patients who recovered did so within 52 hours after the last suppository. Only one patient showed sequelae; he was mentally retarded. There were 4 patients who ran high temperatures and had convulsions, despite therapy. All 4 eventually succumbed within 20 hours following administration of the last suppository. At autopsy, esophageal ulceration with perforation was observed in 2 cases and pulmonary inflammatory changes were present in all cases. Lesions were also present in the liver and brain. The author concluded that rectal suppositories are a poor means of administering theophylline or aminophylline since the unpredictability of absorption makes a fixed therapeutic regimen dangerous. M. C.

Spontaneous Pneumothorax, Mediastinal and Subcutaneous Emphysema in the Asthmatic Patient. Turiaf, J., Marland, P., and Mathieu, H.: Presse méd. **64**: 125, 1956.

The case histories of 2,000 adult asthmatic patients were analyzed. Three developed mediastinal and subcutaneous emphysema. Twelve had spontaneous pneumothorax which was associated with subcutaneous emphysema in two patients. Three of the 15 patients were males. Most of the accidents occurred at the age of 20 to 30 years and usually followed a severe attack of paroxysmal asthma. The most important symptoms were pain in the chest and aggravation of the dyspnea. The displacement of the point of maximal intensity of the heartbeat was the most important and yet inconsistent physical sign. Systemic signs were usually absent. The diagnosis was made roentgenologically.

Generalized pneumothorax occurred usually on the left side and resorbed within 15 to 20 days. One patient with unilateral pneumothorax died. Only rarely was the lung retracted toward the mediastinum. There were 2 cases of unilateral and 2 of bilateral localized pneumothorax, usually involving the apices. These resorbed in a few days or weeks.

Pleural effusion was noted in 4 cases. It was usually minimal and lasted from 6 to 10 days. The fluid was rich in eosinophiles in contrast to the moderate blood eosinophilia. Mediastinal emphysema was seen in the younger

patients and disappeared within a few days. In 6 patients with pneumothorax, emphysematous bullae were demonstrated by x-ray in 4, by pleuroscopy in one, and at post mortem in one. Treatment was directed against the asthma alone. Only one patient received ACTH. Two patients died, and in one of them the chief cause of death was not the pneumothorax. I. G.

Treatment of Asthma With Prednisone. Vallery-Radot, P.: Presse méd. **64:** 273, 1956.

Sixteen severe asthmatics, aged 31 to 77 years, were treated orally with prednisone. Twelve had had constant dyspnea for some days or weeks. Many were cyanotic and 3 were in heart failure, requiring digitalization. Three had severe paroxysmal asthma. One had been treated for 3 years with alternating courses of ACTH, cortisone, and hydrocortisone. The initial dose of prednisone was 25 to 60 mg. in 24 hours, divided into 4 to 6 parts. This was continued until the dyspnea was completely relieved and then the dose was lowered. In less than 3 days, paroxysmal attacks disappeared in all the patients. Except for one case, a complete suppression of dyspnea and cough was achieved within 6 days (in 8 cases, within 48 hours). The duration of action of prednisone appeared to be shorter than that of ACTH and cortisone. Attacks recurred after cessation of therapy. Only one patient had a remission lasting 2 months. One patient who had received alternating ACTH and cortisone courses of 50 to 100 mg. daily for 3 years stated that 15 mg. of prednisone daily was definitely more effective. In all the patients, the effect of prednisone was equal and sometimes superior to that of ACTH, cortisone, or hydrocortisone. Patients who were not relieved by ACTH (1 case), cortisone (1 case), or hydrocortisone (3 cases) reacted well to prednisone.

The author suggests that prolonged treatment with prednisone should be interrupted once in 2 or 3 weeks by 2 injections of 40 units of ACTH gel in order to prevent aplasia of the suprarenal cortex. In 7 patients, an increased diuresis was noted on the third to the seventh day of treatment. Two patients with evident congestive heart failure were rapidly relieved. In 5 out of the 7 bloods examined, there was some lowering of potassium. It is suggested that 2 Gm. of potassium chloride be given daily, especially during maintenance treatment. There were no other electrolyte changes and no glycosuria. Some patients developed Cushing-like facies. No complications or infections were noted. I. G.

Effect of Cortisone on the Pulmonary Cholinergic Hypersensitivity of the Asthmatic. Tiffeneau, R., and Dunoyer, P.: Presse méd. **64:** 719, 1956.

Twenty-four asthmatic patients were treated with 40 to 80 mg. of oral hydrocortisone daily for 4 to 10 days. The pulmonary acetylcholine hypersusceptibility was estimated by measuring the smallest dose of acetylcholine (liminal dose) capable of producing a tangible ventilatory effect when administered by aerosol. The clinical improvement produced by cortisone was not associated with a reduction in the lung sensitivity to acetylcholine. After the administration of adrenalin, the liminal dose of acetylcholine changed from several micrograms to many thousands of micrograms (normal or subnormal values). It is suggested that cortisone does not modify the pulmonary sensitivity to chemical mediators, but acts by inhibiting their formation or release. Adrenalin, on the other hand, reduces the pulmonary sensitivity to acetylcholine and histamine and can bring it back to normal. I. G.

Miscellaneous Allergies

Thrombocytopenic Purpura Due to Quinidine. I. Clinical Studies. Bolton, F. G., and Dameshek, W.: Blood **11**: 527, 1956.

The authors discussed five cases of thrombocytopenic purpura due to quinidine and reviewed all the quinidine-produced thrombocytopenic purpura cases previously described in the literature. The first suggestive case of quinidine purpura was reported by Seyderheim in 1927, but the first unquestionable case was discussed by Broch in 1940. Approximately twenty-three patients had previously been shown to have a thrombocytopenic response to quinidine prior to the authors' report. Of the five cases reported, four occurred in men; this trend was also observed in the previously documented cases. All the patients were over 50 years of age, which was to be expected since this drug is rarely used in younger people. Two of the five patients gave a suggestive family history of allergy.

Platelet studies made on each patient at the beginning of the bleeding episodes revealed counts ranging from 4,000 to 67,000, which quickly rose to normal levels within one to three days after the quinidine was discontinued. Two of the patients also had a mild leukopenia, but in each the blood count quickly returned to normal when the drug was withdrawn. After the reaction had subsided, one of the patients was given a test dose of 0.3 Gm. of quinidine sulphate. Within six hours, the platelet count dropped from 363,000 to 10,000. The patient's tourniquet test became positive and the patient had minimal signs of purpura. The clinical symptoms cleared within twenty-four hours and shortly thereafter the platelet count again was normal. Each patient had had several courses of quinidine before sensitivity became apparent. In the study of previously reported cases, there was no correlation between the amount or the number of times the drug had been taken before the reaction occurred.

Platelet-free plasma from each of these patients agglutinated the platelets of normal platelet-rich plasma in the presence of quinidine, but not in the absence of quinidine. It was also possible to demonstrate that complement fixation occurred during the immunologic reaction. The serum from a patient who had previously taken considerable quantities of quinine as well as quinidine caused agglutination of platelets in the presence of quinine or quinidine. This patient had no clinical hypersensitivity to quinine. Bone marrow studies performed at the height of the reaction revealed that the megakaryocytes were present in normal numbers, but that no platelet production was occurring.

In conclusion, the authors mention that intraoral hemorrhagic bullous lesions should suggest the possibility of a drug thrombocytopenic purpura. When the cause is quinidine, the prognosis is excellent if the drug is stopped immediately. The value of serologic tests in establishing the diagnosis is stressed. R. W.

An Emergency Set to Combat Anaphylaxis. Prickman, L. E., and Lofgren, K. A.: J. A. M. A. **161**: 1159, 1956.

An emergency set for the immediate treatment of anaphylactic reactions should contain the following items: Two 1-c.c. ampules of epinephrine 1:1,000, two 2-c.c. syringes, two hypodermic needles, two long needles (1 inch and 4 inches long), two ampules of aminophylline (0.24 Gm. each), 1 liter of 5 per cent dextrose in distilled water, one intravenous set, one 10-c.c. ampule of Benadryl, one bottle of hydrocortisone (dilution to 2 c.c. gives 50 mg. per c.c.), one ampule of sterile water, one scalpel, one hemostat, one ampule of surgical suture with needle, alcohol, gauze, sponges, swab tongue blade, and one 20-c.c. syringe. M. C.

Anaphylaxis to Heparin Sodium. Report of a Case, With Immunologic Studies. Bernstein, L. I.: J. A. M. A. **161**: 1379, 1956.

A 71-year-old man with myocardial infarction and a long-standing history of seasonal rhinitis and bronchial asthma received 100 mg. of beef heparin on admission to the hospital. A second intravenous injection of 50 mg. of heparin was given 2 weeks later for thrombophlebitis. Within 3 minutes, the patient became dyspneic, cyanotic, and appeared to be in shock. He was given 0.2 c.c. of epinephrine 1:1,000 intravenously and 0.3 c.c. subcutaneously and 50 mg. of diphenhydramine (Benadryl) intravenously. Gradual recovery followed in one-half hour, with no harmful effect on the patient's cardiac status.

Positive reactions were obtained with beef heparin diluted 1:100 on direct testing and by the passive transfer method. Pork heparin, beef muscle, beef serum, pork muscle, and penicillin gave negative reactions, which indicated that the allergic reaction was not caused by animal protein or by other contaminants. The specific antigen was considered to be a heparin polysaccharide fraction which is present in beef heparin but is not present in pork heparin.

M. C.

A Study of the Sensitizing Potential of Novobiocin. Welch, H., Lewis, C. N., Putnam, L. E., and Randall, W. A.: Antibiotic Med. **3**: 27, 1956.

Four groups, each consisting of 105 male volunteers from a penal institution, were selected at random for this study. Eighty-two per cent of the volunteers were Negroes. One group was given one brand of novobiocin; another group was given a second brand of this antibiotic; the third group was given penicillin; and the last group received lactose as a control. The drugs were administered orally in 0.5 Gm. doses twice daily for 5 days. After a rest period of two days, the 5-day course was repeated.

Three subjects treated with novobiocin exhibited yellowing of the sclera. This appeared within 2 days in one subject and within 3 days in the other 2 subjects. Skin tests with novobiocin in these individuals gave negative reactions.

Various types of skin eruptions developed during the administration of novobiocin in 4 subjects. In only one case was the eruption considered to be a drug reaction. In this subject, a maculopapular rash appeared over the chest and arms on the ninth day of administration of this drug. This eruption disappeared three days after the last dose of novobiocin was given. Mildly positive reactions were obtained in this case when the subject was skin tested with 10 mg. of a highly purified novobiocin, with dihydronovobiocin, and with the two preparations of novobiocin used in the study. Control tests with water gave negative reactions. Skin tests performed with novobiocin in the other subjects with skin rashes also gave negative reactions. One of the subjects receiving penicillin developed an eruption. Skin tests performed with 10 units of penicillin gave no reaction in this case.

Loose stools developed in 37 per cent of the volunteers receiving novobiocin. It was not necessary to stop the drug in these cases. Twenty-two per cent of the penicillin-treated group and 7 per cent of the placebo-treated group also developed loose stools.

The authors suggest that the relatively high percentage of skin rashes previously reported following the use of novobiocin is related to excessive dosage. It is proposed that doses of 0.5 Gm. twice daily should be sufficient to control most susceptible infections and with this dosage, skin rashes can be kept at a minimum.

B. S.

Novobiocin (Streptonivicin, Cathomycin): Further Observations. Martin, W. J., Heilman, F. R., Nicols, D. R., Wellman, W. E., and Geraci, J. E.: Proc. Staff Meet., Mayo Clin. **31**: 416, 1956.

Novobiocin was given to 34 patients with infections due to various bacteria. Excellent results were obtained in the treatment of *M. pyogenes* infections. It was suggested that novobiocin may prove to be a special boon in the treatment of resistant strains of this organism. It was considered unlikely that the use of novobiocin will be complicated by micrococcal enterocolitis, since the agent was unable to eradicate *E. coli, A. aerogenes* and *Pseudomonas aeruginosa* from the gastrointestinal tract. The most frequently encountered untoward reaction to the drug was allergic dermatitis, consisting of urticaria and angioedema, which occurred in 5 of the cases. Mild gastrointestinal irritation, nausea, and transient leukopenia also occurred but did not require withdrawal of the agent. M. C.

Death After Bee Sting and Benadryl. Glazer, M.: Harefuah **50**: 172, 1956.

A 20-month-old boy with a negative personal and family history for allergic disease was examined one-half hour after being stung by bees. There was swelling at the sites of the ten bites. He was given 100 mg. of Sevenal and 25 mg. of Benadryl three times daily. After three doses, the Benadryl was discontinued because the child refused to eat and vomited. He was hospitalized on the third day because of almost continuous somnolence. On examination, he was pale yellow in color, indifferent, and reacted slowly to stimuli. The liver was 5 cm. and the spleen 1 cm. below the costal margin. There was a questionable slight nuchal rigidity. A lumbar puncture revealed normal spinal fluid values. On the fourth day, oliguria, jaundice, and a blood pressure of 140/80 were noted. The blood revealed bilirubin, 8.9 mg. per cent (5.8 direct and 3.1 indirect), and urea, 192 mg. per cent. The Takata-Ara test was negative, the thymol flocculation test was positive, and the thymol turbidity was 8 to 9. In the urine, there were traces of albumin, 1 to 3 leukocytes, 5 to 8 erythrocytes, normal urinobilinogen, and no bilirubin. On the seventh day, edema of the face and right hand appeared, and this became generalized before death on the next day. A limited post-mortem examination was permitted; one kidney and part of the liver disclosed changes of lower nephron nephrosis and signs of degeneration and necrosis of liver cells. After considering the possibility that pre-existing disease or intoxication might have affected the liver and kidneys, the author arrived at the conclusion that death had been caused by the Benadryl. I. G.

Anaphylaxis

The Effect of Egg Yolk in Diets on Anaphylactic Arthritis (Passive Arthus Phenomenon) in the Guinea Pig. Coburn, A. F., Graham, C. E., and Haninger, J.: J. Exper. Med. **100**: 425, 1954.

Guinea pigs were kept on one of several diets not only from birth, but prenatally by control of the mothers' diets. Passive Arthus arthritis reactions were induced in these animals, using either an egg albumin–antiegg albumin system or a horse serum–antihorse serum system. Similar results were obtained with both antigens. Guinea pigs born and raised on a diet containing whole egg yolk powder showed about one-half as much joint swelling as guinea pigs raised on a regular stock diet. Histologic studies and serum titrations for a substance which reacts with diphenylamine (DPA) confirmed the protective effect of whole egg yolk. Similar studies carried out with fractions of egg yolk indicated that the protective material was in the alcohol-soluble fraction. Lecithin and sphingomyelin added to the stock diets pro-

tected against the joint swelling but had no effect on the DPA reaction. Further breakdown of crude lecithin yielded substances which inhibited both the passive Arthus arthritis reaction and the DPA phenomenon. These substances could not be identified with any known lipid. E. K.

Anaphylactic Shock in Guinea Pigs Sensitized to Polytyrosylgelatin. Sela, M., Katchalski, E., and Olitzki, A. L.: Science **123**: 1129, 1956.

Gelatin was modified by the attachment of L-tyrosine polypeptides to the free amino groups of gelatin through peptide bands. The polytyrosylgelatin thus obtained contained 16 per cent tyrosine. Guinea pigs were given three intra-abdominal injections at 3-day intervals with polytyrosylgelatin, or gelatin, or with a copolymer of L-aspartic acid and L-tyrosine. Fifteen days after sensitization, an intracardiac injection of one of these substances was administered. Three of 5 guinea pigs sensitized with polytyrosylgelatin showed typical anaphylactic shock and died after an intracardiac injection of the same substance. The other two animals exhibited marked diminution in body temperature. The gelatin and the copolymer of L-aspartic acid and L-tyrosine failed to show evidence of antigenic activity. It was concluded that the attachment of tyrosine polypeptides enhances the antigenicity of gelatin. B. S.

Effects of Anaphylactic Shock and Compound 48/80 on the Mast Cells of the Guinea Pig Lung. Mota, I., and Vugman, I.: Nature **177**: 427, 1956.

Experiments were carried out on several groups of guinea pigs. Soon after death, the lungs were fixed by injecting the fixative through the trachea. Frozen sections of the lung, 50 μ. thick, were stained with toluidine blue in acidified solution and the number of mast cells was determined. In animals sensitized with egg albumin and shocked three weeks later by intracardiac injection of the same antigen, there was a significant reduction in the number mast cells, degranulation, and decrease of metachromasia of the remaining granules in most of the cells. In another group of sensitized animals protected with antihistamine one hour before the injection of the shocking dose and killed two hours after it, the mast cells presented the same alterations. It appeared that the antihistamine was ineffective in protecting mast cells from the damage resulting from the specific antigen but acted by blocking tissue receptors against free histamine. The injection of 0.5 mg. of compound 48/80 intracardially produced a clinical picture similar to that presented by the sensitized animals injected with the antigen. However, no morphologic or numerical changes could be found in the mast cells of these guinea pigs. No changes were found in animals killed by intracardiac injection of histamine, by a blow on the head, or by jugular bleeding. The experiments suggested that the mast cells were the source of histamine liberated during anaphylactic shock in guinea pigs. I. G.

Pharmacology, Physiology, and Pathology

The Inhibiting Effect of Epinephrine and Norepinephrine on Secretion Induced by Histamine in Separated Pouches of Dogs. Forrest, A. P. M., and Code, C. F.: J. Pharmacol. & Exper. Therap. **110**: 447, 1954.

Dogs with separated stomach pouches were injected intravenously with epinephrine or norepinephrine over a period of time. Histamine was also administered by continuous intravenous injection. Both epinephrine and norepinephrine strongly inhibited the volume of histamine-induced gastric secretion. The rapidity of onset and of recovery of the inhibition suggests that the effect on gastric secretion may be of vascular origin. These inhibitors effected little or no change in hydrochloric acid concentration. E. K.

The Chemical Measurement of Histamine in Blood Plasma and Cells. Lowry, O. H., Graham, H. T., Harris, F. B., Priebat, M. K., Marks, A. R., and Bregman, R. U.: J. Pharmacol. & Exper. Therap. **112**: 116, 1954.

A method is described for the measurement of histamine in plasma and cells which permits the estimation of 0.01 to 0.02 micrograms histamine from plasma concentrations which originally were as low as 4×10^{-10} (3×10^{-9M}) with 25 per cent precision. Histamine is extracted with trichloroacetic acid, concentrated and separated from other amines by adsorption, converted to a colored product by combination with dinitrofluorobenzene (DNFB), further separated from other amines by extraction with methyl-n-hexyl ketone and hydrochloric acid, and finally subjected to spectrophotometric measurement. A variety of other amines were checked for degree of interference. Many were eliminated either by failure to be absorbed on Decalso or by failure to hold DNFB color through the extraction process. Histaminase controls were also done. Added histamine was totally destroyed by this enzyme. Blood samples from 54 individuals were analyzed. The average histamine level of plasma computed by this method was 4.3 micrograms per liter, of which 60 per cent was destroyed by histaminase. Thus, true plasma histamine is less than 3 micrograms per liter. The buffy coat contains 2,000 times as much histamine as plasma. "However, as reported earlier . . . the histamine is practically confined to the neutrophilic and basophilic granulocytes. (The basophils contain about 500,000 microgm. per kgm.)" Red blood cells contain interfering substances which are not destroyed by histaminase, but "in any event seem to contain quantitatively insignificant amounts of histamine." It is suggested that the measurement of histamine in whole blood is not worth while, as it "would merely reflect the neutrophil and basophil count." Plasma levels, on the other hand, may prove significant. E. K.

Antihistaminics as Epinephrine Sensitizers. Kuriaki, K., and Uchida, T.: J. Pharmacol. & Exper. Therap. **115**: 228, 1955.

Benadryl, Diatrin, and Anergen were perfused into isolated toad hearts, toad hindlegs, and rabbit intestinal strips. They were also administered to anesthetized rabbits and cats in order to study their effects on blood pressure, respiration, and response of the nictitating membrane. The technique employed was as follows: Epinephrine was added, then there was a recovery period; the antihistaminic was added and, within a few minutes of this, epinephrine was again administered. Benadryl and Anergen enhanced the response of peripheral organs to epinephrine and to sympathetic stimulation. These drugs also inhibited amine oxidase activity. Diatrin failed to inhibit amine oxidase and showed no consistent effects on epinephrine responses. All three drugs blocked the vasodepressor effects of electric vagal stimulation. E. K.

Dilator Responses to Isoproterenol in Cutaneous and Skeletal Muscle Vascular Beds: Effects of Adrenergic Blocking Drugs. Walters, P. A., Jr., Cooper, T. W., Denison, A. B., Jr., and Green, H. D.: J. Pharmacol. & Exper. Therap. **113**: 323, 1955.

An electromagnetic flowmeter was used to record rates of blood flow in cutaneous and skeletal muscle vascular beds of anesthetized dogs. Isuprel, Levophed, and Adrenalin were injected intra-arterially before and after the administration of the sympathomimetic blocking agents, Ilidar or Dibenzyline. In skeletal muscle, the initial response to Adrenalin and Levophed was vasoconstriction. Dibenzyline abolished the constrictive phase of Adrenalin activity

and unmasked the vasodilation component. No vasodilator response was observed when the vasoconstrictive phase of response to Levophed was thus abolished. Even Isuprel responses initially consisted of vasodilation. Large doses of blocking agent, comparable to those which blocked Adrenalin dilator responses, also blocked Isuprel vasodilation. In the skin, similar responses were noted except that the blocking agents failed to unmask any dilator response to Adrenalin, and Isuprel vasodilation was not as easily inhibited by the blocking agents. E. K.

Skin Tests With Bacterial Antigens in Rheumatoid Arthritis. Swineford, O., Jr., Coleman, W. P., and Hyde, A. T., Jr.: Ann. Allergy **14**: 139, 1956.

Patients suffering from rheumatoid arthritis were tested with the following substances: (1) bacterial vaccines; (2) polysaccharide and nucleoprotein fractions of 12 bacterial species; (3) broth culture filtrates of microorganisms; and (4) nonbacterial antigens. A total of 2,164 skin tests were performed with the vaccines and nonbacterial allergens. Approximately 38 per cent of these tests yielded positive reactions of the immediate type, and 45 per cent of them gave reactions of the delayed type. There were 917 skin tests performed with mixtures of crude polysaccharide and nucleoprotein fractions of several strains of bacteria, and 1,035 tests were performed with mixtures of filtrates of broth cultures of various bacterial strains. Large numbers of positive reactions were obtained to all these tests, with no real preponderance of reactions to any one antigen. The reactions followed no pattern and there was no indication that the antigens eliciting the strongest reactions were of etiological significance. Sixty-four patients were treated with an arbitrary mixture of the bacterial antigens to which many positive reactions had been obtained. Five of these patients showed improvement, while three patients obtained marked and lasting remission of symptoms following treatment with the mixed antigen. The skin reactions manifested by those patients benefited by treatment did not differ from the skin reactions manifested by those patients not benefited. These results, along with findings of other workers, suggest that "at least some rheumatoid arthritis cases are the result of a delayed-type of antigen-antibody reaction which may be initiated by a variety of antigens." H. F.

Effect of Cortisone on the Serum Gamma-Globulin. Snell, R. S., and Nicol, T.: Nature **177**: 578, 1956.

Twelve guinea pigs were given daily doses of 10 mg. of cortisone intramuscularly for five weeks. The gamma-globulin fraction of serum protein of cardiac blood was estimated at weekly intervals and taken as a measure of the antibody level. It was estimated by first separating the gamma-globulin fraction by paper electrophoresis, then treating the paper with dyes, and estimating the protein dye complex by means of a photoelectric cell. In all the animals the gamma-globulin level became markedly reduced during the first two weeks of cortisone treatment, and it remained low in all but two.
I. G.

The Influence of ACTH, Cortisone, and Hydrocortisone on the Distribution and Peripheral Metabolism of Thyroxine. Ingbar, S. H., and Freinkel, N.: J. Clin. Invest. **34**: 1375, 1955.

Prolonged administration of cortisone or ACTH may produce a clinical picture of hypothyroidism and may have a pronounced effect on iodine metab-

olism. Eight myxedematous patients were adequately treated with thyroid hormone. They showed normal values for rate of peripheral degradation of thyroid hormone as measured by a radioactive thyroxine test. Serial measurements of serum-precipitable iodine were also made. Two patients with moderate hypothyroidism and panhypopituitarism who were not specifically treated for their deficiencies were studied in addition. Cortisone, hydrocortisone, or ACTH was administered in various doses, and after 4 to 5 days of treatment thyroxine degradation studies were repeated. There was no evidence that ACTH or adrenal steroids altered the rate of degradation of thyroxine. The adrenal hormones did reduce the volume of distribution of thyroid hormone and increased the fractional rate of turnover of thyroxine. These two effects balanced each other so that the daily volume turnover was unchanged. The total quantity of thyroxine in extrathyroidal tissues decreased. It is suggested that the reduction in concentration of circulating thyroid hormone observed following administration of adrenal hormones may be due either to diminution of glandular production of thyroid hormone, to a decline in the rate of turnover of the glandular hormone pool, or to both. E. K.

Properdin and Its Significance in Immunology. Present Status of the Question. Delaunay, A., Henon, M., Bazin, S., and Pelletier, M.: Presse méd. **64**: 65, 1956.

Zymosan (Z), a soluble residue obtained by treating fresh yeast with trypsin and alcohol, when added to human serum at certain temperatures, unites with a particular factor of the serum named properdin (P) to form an insoluble complex (PZ) which inactivates the third constituent of complement (C'_3). PZ has little effect on other constituents of complement, thus differing from typical antigen-antibody reactions. Other substances, such as complex hydrocarbons, some dextrans and levans, cell membranes of numerous bacteria, and debris of red cells, are also capable of "fixing" properdin and secondarily inactivate C'_3. Properdin is particularly high in the serum of rats, low in the serum of guinea pigs, and average in the serum of human beings. In general, it is higher in animals which have a high natural immunity to bacteria and viruses. Properdin is absent in cerebrospinal fluid, exudates, pleural and peritoneal transudates, colostrum, milk, and extracts of leukocytes and platelets. It can be prepared from human and bovine sera and titrated by its ability to inactivate C'_3 in the presence of zymosan when added to serum from which properdin has been removed (RP). Human properdin is an euglobulin of a molecular weight of at least 8 times that of the γ-globulins. It does not represent more than 0.03 per cent of the total serum proteins. It is not inactivated or fixed by antigen-antibody aggregates and is not necessary for the classical immunologic reactions. In vitro, properdin, associated with complement in the presence of Mg^{++} (the so-called properdin system), can cause hemolysis of certain abnormal erythrocytes, such as those of patients with nocturnal paroxysmal hemoglobinuria, and of normal erythrocytes treated with dilute tannic acid. Properdin has a bactericidal action. It is apparently involved in the thermolabile factor of human and animal serum which is capable of inactivating some viruses. Properdin increases the resistance of animals to the harmful effect of radiation and bacterial infection. I. G.

The Release of Histamine and 5-Hydroxytryptamine (Serotonin) From Platelets by Antigen-Antibody Reactions (In Vitro). Humphrey, J. H., and Jaques, R.: J. Physiol. **128**: 9, 1955.

Rabbits were given either single or multiple courses of intravenous injections of formalin-killed pneumonococci in order to prepare antisera against

the purified capsular polysaccharide of the Type III pneumonococcus. Other rabbits were sensitized with 5 times recrystallized ovalbumin administered by two courses of intravenous injections of alum-precipitated hen ovalbumin. From these sera, the gamma globulin fractions were precipitated out by means of a salting process and stored freeze-dried. The antibodies were considered to be purified, but, in each case, only one-third of the material was specifically precipitable with the corresponding antigen.

The addition of antigen to rabbit platelets suspended in heparinized plasma which contained specific antibody was followed by release from the platelets of histamine and 5-hydroxytryptamine. The two substances were released simultaneously. "Direct microscopical observation by phase-contrast illumination, using silicone-treated slides and cover-slips, at 37° C., did not reveal any obvious changes in platelet morphology when antigen solution was allowed to mix with a plasma-antibody-platelet mixture, although the platelets became sticky and tended to clump." The presence of plasma or serum and of free calcium ions was necessary for the reaction. The addition of antigen and antibody to either heparinized serum or plasma caused a similar release. Since the serum contained no fibrinogen, fibrin formation was not involved in the mechanism of histamine release. Heating the plasma at 56° C. for 30 minutes greatly lessened its activity, suggesting that complement was probably active in the reaction. Since the release resulting from the antigen-antibody combination occurred in the presence of heparin concentrations sufficient to inhibit the activity of serum, the mechanism of release did not involve thrombin. Rabbit plasma in which antigen-antibody reactions had occurred did not release histamine from platelets or show proteolytic activity when tested one-half hour or more later. suggesting that the activation of plasma toward platelets was transient. Similar liberation of histamine and/or 5-hydroxytryptamine from homologous platelets by antigen-antibody reactions in human, dog, and guinea pig plasmas was also observed.

When circulating platelets were removed from the blood stream of guinea pigs, no difference was noted between the passive anaphylactic response to rabbit antipneumonococcus globulin by these animals and by normal guinea pigs. The authors concluded that anaphylaxis in guinea pigs is not dependent on the presence of platelets.

The authors suggest that the general features of the reaction indicate an enzymic mechanism for release rather than a mechanical entanglement of the platelets followed by disruption. They concluded that the blood clotting mechanism was not directly connected with the release brought about by the antigen-antibody combination. The authors outlined the following possible sequence of events: "antigen-antibody combination $\xrightarrow{CA''}$ fixation of some component(s) of complement, followed by activation of protease." R. W.

Antibody Formation in the Omentum. Roberts, K. B.: Brit. J. Exper. Path. **36**: 357, 1955.

Rabbits were injected intraperitoneally or intravenously with killed *Salmonella typhi* organisms one or more times. Serum and tissue agglutinating antibody titers were determined at varying intervals after the last injection of antigen. A high level of antibody was found in the omentum when the intraperitoneal route was employed, but was not found when the intravenous route was used. Studies done several weeks after a single intraperitoneal injection, when the acute inflammatory reaction in the omentum had subsided, revealed a higher level of antibody in this organ than in the kidney, liver, or lymph nodes. In another experiment, *Salmonella typhi* was injected intravenously simultaneously with an intraperitoneal injection of *Salmonella paratyphi B.* In each animal, the omentum showed a higher tissue titer than other organs

to *Salmonella paratyphi B*, but a relatively low tissue titer to *Salmonella typhi*. These results are consistent with the premise that the omentum is involved in the production of antibody after an intraperitoneal injection of antigen.

In another experiment, an intraperitoneally sensitized rabbit was sacrificed and the omentum was removed. One part was transferred immediately into the peritoneal cavity of a normal animal. The other part was heated to 56° C. for 30 minutes before being transferred to the peritoneal cavity of another normal animal. The heating process destroyed all cells in the omentum, but did not affect the antigen or antibody. It was found that serum agglutinin titers were consistently higher in the animal receiving living omental tissue.

Histologic studies of the omentum at varying periods after an intraperitoneal injection revealed an early polymorphonuclear leucocyte response. However, after 5 days, there was superimposed on the inflammatory response a new formation of pyronine-staining cells. These appeared in the subserosal tissues, especially in the hypertrophied milk spots. The majority of the pyronine-staining cells were typical plasma cells. B. S.

Miscellaneous

A Biological Factor Inhibiting the Effect of Cortisone on Antibody Formation. Berglund, K., and Fagraeus, A.: Nature **177**: 233, 1956.

The production of hemolysin in the rat following an intraperitoneal injection of sheep erythrocytes was investigated. Cortisone was given intramuscularly daily for six days, beginning four days before the antigen was injected. The cortisone produced a significant reduction of the hemolysin response. When a suspension of rat spleen cells was administered twice daily intraperitoneally for four days beginning five hours after the antigen injection, the mean antibody titer was significantly enhanced and there was no depression of the antibody response. The same result was achieved by administering only one injection of spleen cells on the day of the antigen injection. A suspension of rat thymus cells had a similar effect. The injection of spleen cells on the first, second, or third day after the antigen injection had not been significantly effective. When given one day before the antigen, it did not protect the antibody production against cortisone. Distintegration of the spleen cells (grinding in a homogenizer at high speed) destroyed their protective effect on antibody formation.

The authors suggest that "the spleen and thymus cells contain or, more probably, produce a factor, or have a cellular function essential for some initial, cortisone-sensitive phase of the antibody production process." I. G.

Pollen From Leda Clay of Maine. Knox, A. S.: Science **123**: 1118, 1956.

Leda clay (so named because of the presence of species of Leda) from Mount Desert Island in Maine was studied for microfossils. After separation with a bromoform-acetone mixture, a large number of pollen grains were recovered. An analysis of these grains showed 51 per cent spruce, 28 per cent pine, 7 per cent fir, 7 per cent grass, and smaller amounts of elm, oak, hemlock, rose family, and fern spores. The exact age of Leda clay is not known, but it is believed to have been deposited in late glacial time during the marine submergence. On the basis of the fossil plants and pollen found in the clay, it seems probable that extensive growth existed in eastern Maine at that time and that the flora and climate were similar to those of southern Labrador today. B. S.

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